DISSERTATION ON

THE ROLE OF SYSTEMIC CORTICOSTEROID THERAPY IN NON ARTERITIC ANTERIOR ISCHAEMIC OPTIC NEUROPATHY

Submitted in partial fulfillment of requirements of

M.S.OPHTHALMOLOGY

BRANCH – III

REGIONAL INSTITUTE OF OPHTHALMOLOGY

MADRAS MEDICAL COLLEGE

CHENNAI – 600 003

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY,

CHENNAI

APRIL 2016

CERTIFICATE

This is to certify that the dissertation titled "**THE ROLE OF SYSTEMIC CORTICOSTEROID THERAPY IN NON ARTERITIC ANTERIOR ISCHAEMIC OPTIC NEUROPATHY**" is a bonafide record of the research work done by **DR. AKILA.C,** Post graduate in the Regional Institute of Ophthalmology & Government Ophthalmic Hospital, Madras Medical College and Government General Hospital, Chennai-03, in partial fulfilment of the regulations laid down by the Tamil Nadu Dr. M.G.R Medical University for the award of M.S. Ophthalmology Branch III, under my guidance and supervision during the academic year 2013 – 2016.

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I hereby declare the dissertation entitled **"THE ROLE OF SYSTEMIC CORTICOSTEROID THERAPY IN NON ARTERITIC ANTERIOR ISCHAEMIC OPTIC NEUROPATHY"** is a bonafide and genuine research work carried out by me under the guidance of Prof. **Dr. M.ANANDA BABU, M.S, D.O.**

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PLACE:

ACKNOWLEDGEMENT

I express my sincere thanks and gratitude to **Prof. Dr. R. VIMALA, M.D.,** Dean, Madras Medical College, for permitting me to conduct this study.

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Finally I would like to show my gratitude to all the patients and their relatives for participating in this study.

ETHICS COMMITTEE APPROVAL

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CERTIFICATE OF APPROVAL

To
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Dear Dr.Akila C,

The Institutional Ethics Committee has considered your request and approved your study titled **"The role of systemic corticosteroid therapy in non arteritic anterior ischaemic optic neuropathy" No.09062015.**

The following members of Ethics Committee were present in the meeting held on 09.06.2015 conducted at Madras Medical College, Chennai-3.

1.	Prof.C.Rajendran, M.D.,	: Chairperson
2.	Prof.R.Vimala, M.D., Dean, MMC, Ch-3	: Deputy Chairperson
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13.	Tmt.Arnold Saulina, M.A., MSW.,	: Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

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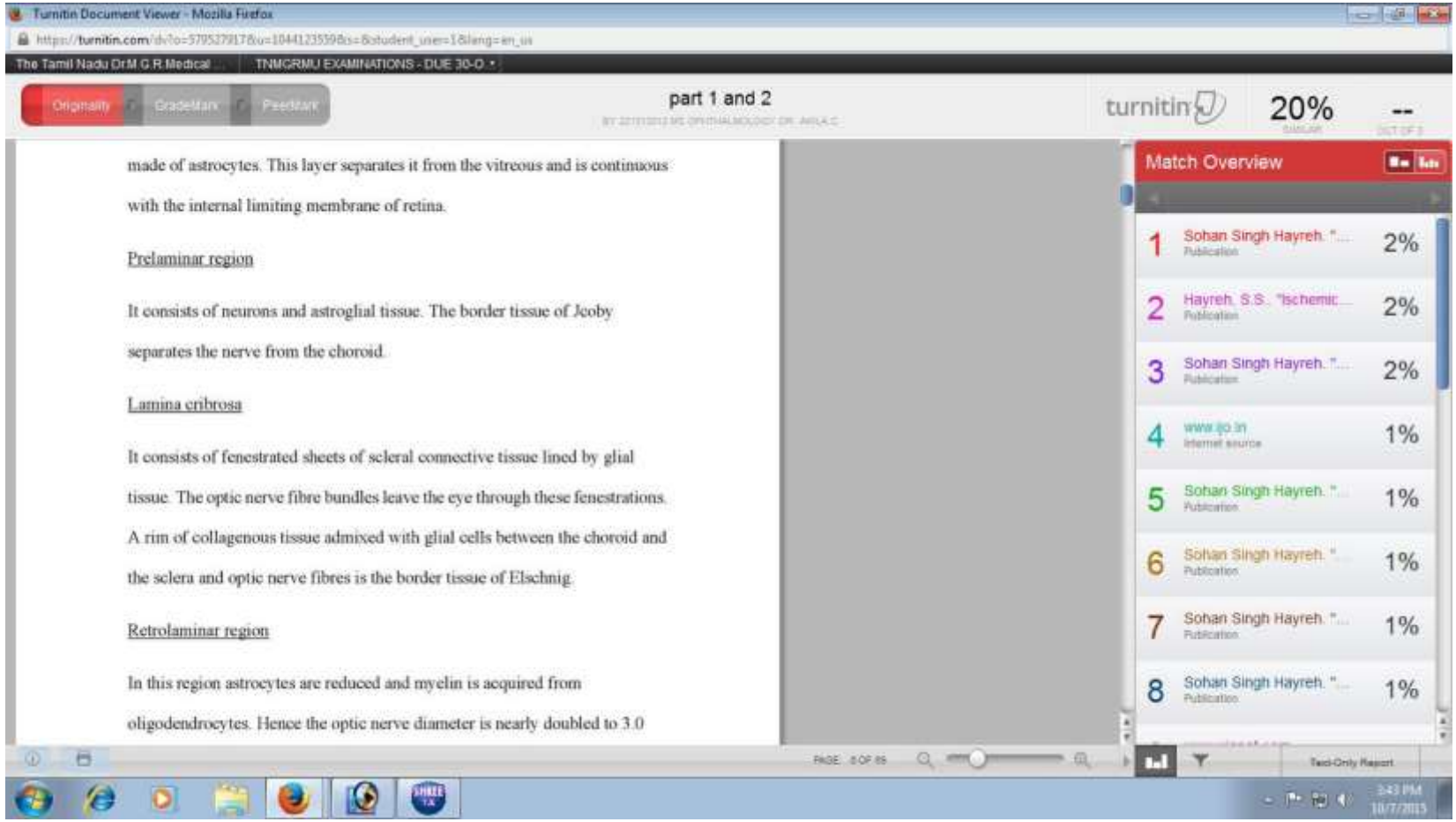
PART I



TURNITIN PLAGIARISM CHECK

CONTENTS

ABSTRACT

The role of systemic corticosteroid therapy in Non Arteritic Anterior Ischaemic Optic Neuropathy

AIM:To look for improvement in visual acuity,visual fields and rate of resolution of optic disc edema in NA-AION after treatment with oral corticosteroids.METHOD:40 patients were diagnosed as NA-AION in acute phase.20 patients among them were randomly selected for steroid therapy with 60mg oral prednisolone once daily for 2 weeks,tapered by 5mg every 5 days to 40mg either till the disc showed no edema or upto a maximum of 2 months and then rapidly tapered off.20 other patients were treated with placebo(oral vitamin C). Visual acuity and visual fields were assessed during completion of therapy & at 6 months.RESULTS:At 6 months from onset of NA-AION,visual acuity improved in 80% in treated group and 30% in control group. Visual fields improved in 40% in treated group and 30% in control group. Optic disc edema resolved earlier in 70% in treated group and 20% in control group.CONCLUSION:Oral corticosteroids in acute phase of NA-AION cause significant improvement in visual acuity, visual fields & rate of resolution of optic disc edema.

Keywords: Non arteritic anterior ischaemic optic neuropathy, corticosteroids, optic disc edmema

PART –I

INTRODUCTION

INTRODUCTION

Ischaemic optic neuropathy is an acute vascular optic neuropathy presenting with sudden “stroke like” vision loss in elderly patients. When the anterior portion of the optic nerve supplied by the posterior ciliary artery circulation is involved, it is Anterior Ischaemic Optic Neuropathy (AION). Posterior Ischaemic Optic Neuropathy (PION) involves that part of the optic nerve which does not receive blood supply from the posterior ciliary arteries. Etiologically, Ischaemic optic neuropathy can be classified as arteritic and non-arteritic types. The most common type of acute optic neuropathy in elderly people is Non Arteritic Anterior Ischaemic Optic Neuropathy (NA-AION) with an annual incidence of 2.3-10.2 per 100000 population.

APPLIED ANATOMY

APPLIED ANATOMY

THE OPTIC NERVE- STRUCTURE

The axons taking origin from the ganglion cells form the retinal nerve fibre layer which continues backward as the optic nerve.

The length of the optic nerve is 47-50 mm. It is divided into four parts:

Intraocular part measures 1 mm

Intraorbital part measures 30 mm

Intracanalicular part measures 6-9 mm

Intracranial part measures 10 mm

INTRAOCULAR PART

Its average horizontal diameter is 1.5 mm and vertical diameter is 1.8 mm. it consists of surface nerve fibre layer, prelaminar portion, lamina cribrosa and retrolaminar portion from anterior to posterior.

Surface nerve fibre layer

It consists of axons originating from the ganglion cells which converge on the optic disc. The optic disc is ensheathed by an internal

limiting membrane made of astrocytes. This layer separates it from the vitreous and is continuous with the internal limiting membrane of retina.

Prelaminar region

It consists of neurons and astroglial tissue. The border tissue of Jcoby separates the nerve from the choroid.

Lamina cribrosa

It consists of fenestrated sheets of scleral connective tissue lined by glial tissue. The optic nerve fibre bundles leave the eye through these fenestrations. A rim of collagenous tissue admixed with glial cells between the choroid and the sclera and optic nerve fibres is the border tissue of Elschnig.

Retrolaminar region

In this region astrocytes are reduced and myelin is acquired from oligodendrocytes. Hence the optic nerve diameter is nearly doubled to 3.0 mm.

INTRAORBITAL PART

The part of the optic nerve from behind the eye to the optic foramina is the intraorbital part. The ophthalmic artery runs above the optic nerve laterally to medially.

INTRACANALICULAR PART

The ophthalmic artery crosses optic nerve from medial to lateral side inferiorly

INTRACRANIAL PART

The cavernous sinus is located below it. This part forms the optic chiasma posteriorly after uniting with its counterpart from the contralateral side. This part is ensheathed by pia mater, but receives arachnoid and dural sheaths at the point of its entry into the optic canal.

BLOOD SUPPLY OF THE OPTIC NERVE

The posterior ciliary arteries with a small contribution from central retinal arteries supply the optic nerve. The anterior and posterior parts of the optic nerve receive blood supply from different sources.

Surface nerve fibre layer:

It is supplied by capillaries derived from the retinal aterioles which anastamose with the vessels from the prelaminar region. Occasionally, a ciliary derived vessel from the prelaminar region may enlarge to form the cilioretinal artery.

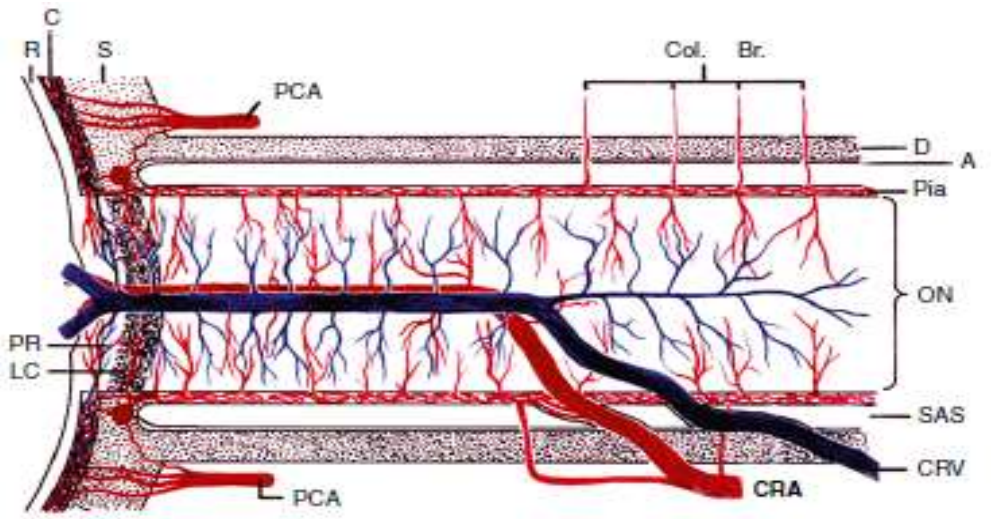


Figure 1 – schematic representation of blood supply of optic nerve

Prelaminar region:

It is supplied by vessels of ciliary region. These vessels are derived from separate branches of short PCAs.

Lamina cribrosa region:

It is also supplied by ciliary vessels. They commonly arise from short PCAs and the arterial anastamosis of Zinn-Haller.

Retrolaminar region:

It is supplied by recurrent pial vessels of the ciliary circulation and centripetal and centrifugal branches of the central retinal artery.

Hence, the major blood supply to the optic nerve head is by the short Posterior Ciliary Arteries and peripapillary choroid.

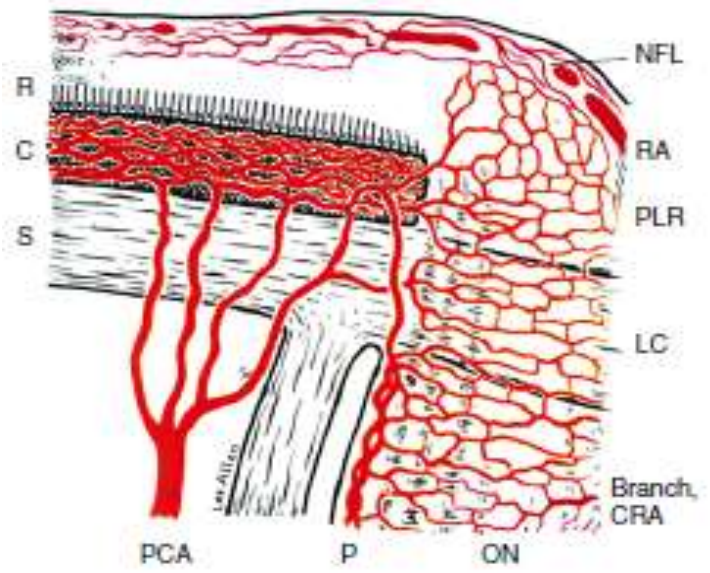


Figure 2- schematic representation of blood supply of nerve fibre layer, prelaminar,laminar and retrolaminar parts of optic nerve

Blood supply of the posterior portion of the optic nerve:

Peripheral centripetal vascular system- it is formed by the pial vessels which come from collateral arteries arising from the ophthalmic artery.

Axial centrifugal vascular system- it is formed by branches arising from the central retinal artery and is seen in 75% of cases.

Posterior ciliary arteries:

There may be 1-5 PCAs but usually 2 or 3 PCAs are present in one eye.

1. Long PCAs- usually medial and lateral arteries - supply the medial and lateral parts of the peripheral choroids. They do not supply the optic nerve head.

2. Short PCAs (SPCAs)- usually upto 20 in number.

    a) Paraoptic SPCAs- few SPCAs enter the eyeball closest to the optic nerve and form the major supply of the optic nerve head.

    b) Distal SPCAs-enter the eyeball midway between the paraoptic SPCAs and long PCAs and form the major supply of the choroids.

The number and blood supply of the PCAs vary from one individual to the other. The PCAs and their subdivisions are endarteries and hence watershed areas are present in the zones of blood supply of the posterior ciliary arteries.The importance of these watershed zones is that when the perfusion pressure drops in any of the endarteries[6], these zones become most vulnerable to ischaemia. This plays an important role in ischaemic optic neuropathy.

The inter- and intra- individual variations in the PCA distribution in blood supply results in varying patterns and manifestations of ischaemic optic neuropathy.

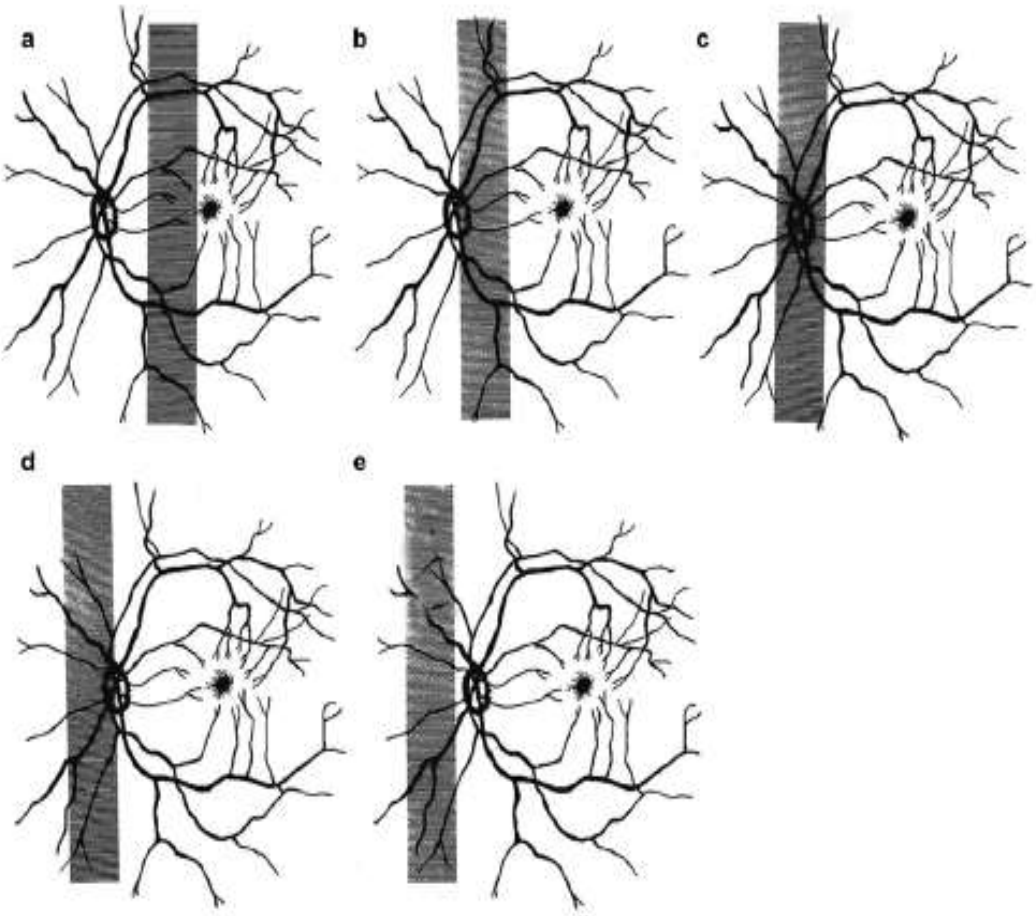


Figure 3- schematic representation of the various locations of watershed zones in optic nerve.

ETIOLOGY AND PATHOGENESIS

ETIOLOGY AND PATHOGENESIS

FACTORS AFFECTING BLOOD FLOW IN THE OPTIC NERVE HEAD AND ITS PATHOPHYSIOLOGY:

Ischaemic optic neuropathy occurs when the blood flow in the optic nerve head is impaired. The blood flow in the optic nerve head is influenced by

Arterial blood pressure,

Intraocular pressure and

Resistance to blood flow.

The ONH blood flow is calculated by the following formula:

Perfusion pressure is the difference between Mean arterial blood pressure and venous blood pressure in a vascular bed. Normally the intraocular pressure is slightly lower than the central retinal venous pressure at the optic disc. Hence, perfusion pressure is the difference between Mean arterial blood pressure and the intraocular pressure[7].

Mean BP = Diastolic BP + 1/3 (systolic minus diastolic BP)

Autoregulation of blood flow and endothelial derived vasoactive agents affect BP and resistance to blood flow.

AUTOREGULATION OF BLOOD FLOW

When changes occur in the perfusion pressure, the blood flow to the tissues and capillary pressure get altered and it is Autoregulation that helps in maintaining a fairly constant blood flow. Autoregulation is stimulated by changes in resistance of the blood vessels which occurs due to alterations in the vascular tone. Autoregulation occurs only when the perfusion pressure is within a particular range.

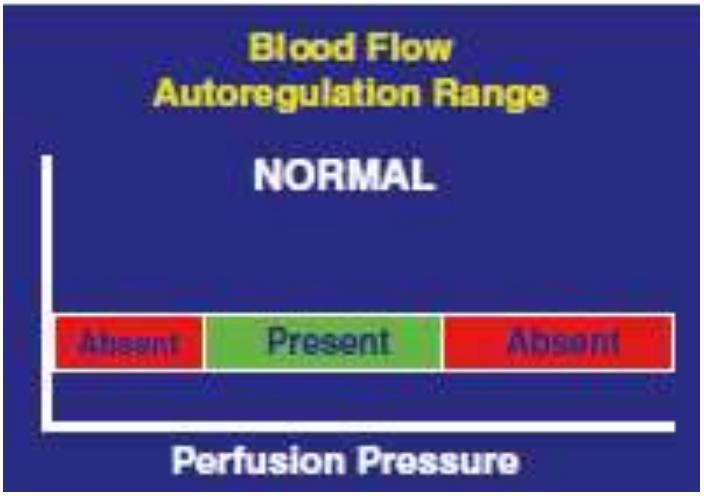


Figure 4- Autoregulation at different perfusion pressure ranges

NA-AION can occur due to impaired autoregulation of the ONH blood flow caused by systemic hypotension.

Autoregulation can be affected by systemic hypertension and hypotension, arteriosclerosis,diabetes,atherosclerosis, hyperlipidemia and vasculitic disorders[11].

ENDOTHELIAL DERIVED VASOACTIVE AGENTS

The endothelial vasodilators like nitric oxide and vasoconstrictors like endothelin control the vascular tone. The autoregulation of blood flow and the vascular tone is also influenced by the endothelial cells[8].

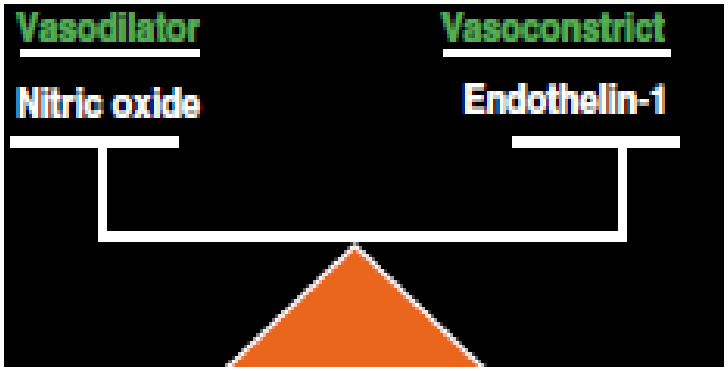


Figure 5- balance between vasodilators and vasoconstrictors

ARTERIAL BLOOD PRESSURE

Arterial hypertension can interfere with the ONH blood flow by causing increased vascular resistance in terminal arterioles or secondary changes in ONH blood flow autoregulation due to abnormalities in Endothelial Derived Vasoactive Agents

INTRAOCULAR PRESSURE

During sleep, rise in IOP and concurrent nocturnal arterial hypotension causes marked fall of perfusion pressure in the ONH. This is an important hidden risk factor for NA-AION[9].

RESISTANCE TO BLOOD FLOW

It is influenced by caliber of the vessels feeding the ONH and rheological properties of the blood.

NON ARTERITIC ANTERIOR ISCHAEMIC OPTIC NEUROPATHY

NA-AION occurs due to vasculopathy involving the para-optic branches of short posterior ciliary arteries but the choroidal circulation is uninvolved. It may be due to transient absence or reduction in perfusion of the vessels or rarely due to embolism of the vessels that supply the ONH. The optic nerve head is supplied mainly by the PCA circulation. But it is not necessary for complete occlusion of the PCA for NA-AION to occur[4]. When the perfusion pressure is reduced, the vessels in the prelaminar portion of the ONH are most susceptible to obliteration followed by peripapillary choroid, watershed zone and rest of the choroid.

RISK FACTORS

PREDISPOSING FACTORS

- Arterial hypertension
- Diabetes mellitus
- Hyperlipidemia
- Ischaemic heart diseas
- Cerebro vascular disease
- Migraine
- Prothrombotic conditions – lupus anticoagulant, ACL antibody, factor 5, protein C & S deficiency, anti thrombin deficiency
- Hyper homocysteinemia
- Drugs – sildenafil, alpha interferon, amiodarone

PRECIPITATING FACTORS

- Nocturnal hypotension
- Sleep apnea

OCULAR FACTORS

- Chronic simple glaucoma
- ONH drusen

COMPARTMENT SYNDROME IN NA-AION

Optic disc in NA-AION is usually of small diameter with a small or absent cup. This causes chronic mechanical obstruction to axoplasmic flow at the level of cribriform plate with resultant intracellular axonal swelling. This axoplasmic stasis causes secondary compression and further microvascular compromise. The decreased return of neurotrophins causes additional ganglion cell death.[5] This compartment syndrome if unrelieved leads to cavernous degeneration of the axons.

CLINICAL FEATURES

CLINICAL FEATURES

SYMPTOMS

Loss of vision in NA-AION occurs over hours to days and more commonly upon awakening. It mostly involves the inferior field of vision. The vision loss is either static or progressive until stabilization.

SIGNS

Visual acuity in NA-AION is more than 6/60 in 58-60% cases. Colour vision loss parallels visual acuity loss.

Visual field defects of any form can occur but altitudinal field defect (usually inferior) is more common and is seen in 55-80% cases.

Optic disc edema is either diffuse or segmental[12], hyperemic or pale. Peripapillary retinal hemorrhages are common. Retinal exudates are unusual. Optic disc in contralateral eye usually has a small diameter with a small or absent cup. This structural crowding of axons is called a “disc at risk”[10].

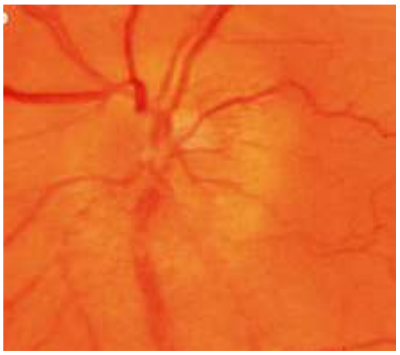


Fig 6- diffuse hyperaemic disc edema in NA-AION

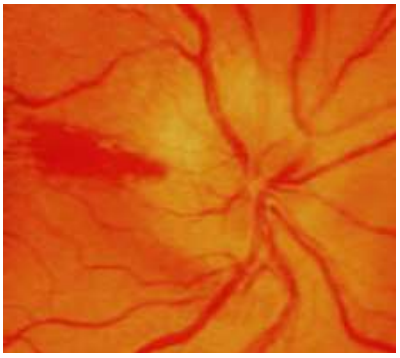


Figure 7- diffuse pallid disc edema with splinter hemorrhage in NA-AION

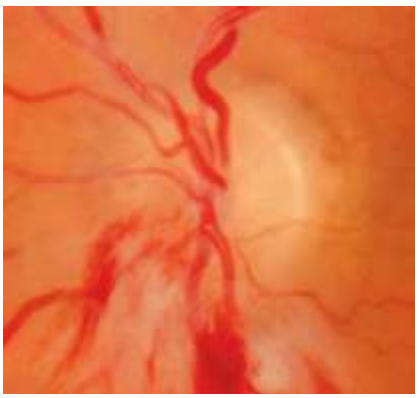


Figure 8- inferior segmental disc edema with splinter hemorrhage in NA-AION

Figure 9- disc pallor after 8 weeks in NA-AION

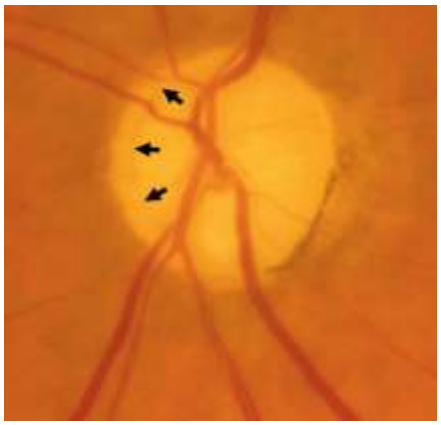


Figure 10- disc pallor with cupping in A-AION

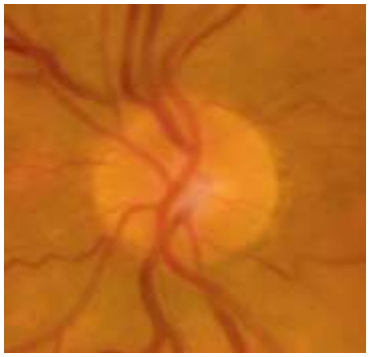


Figure 11- disc at risk in NA-AION

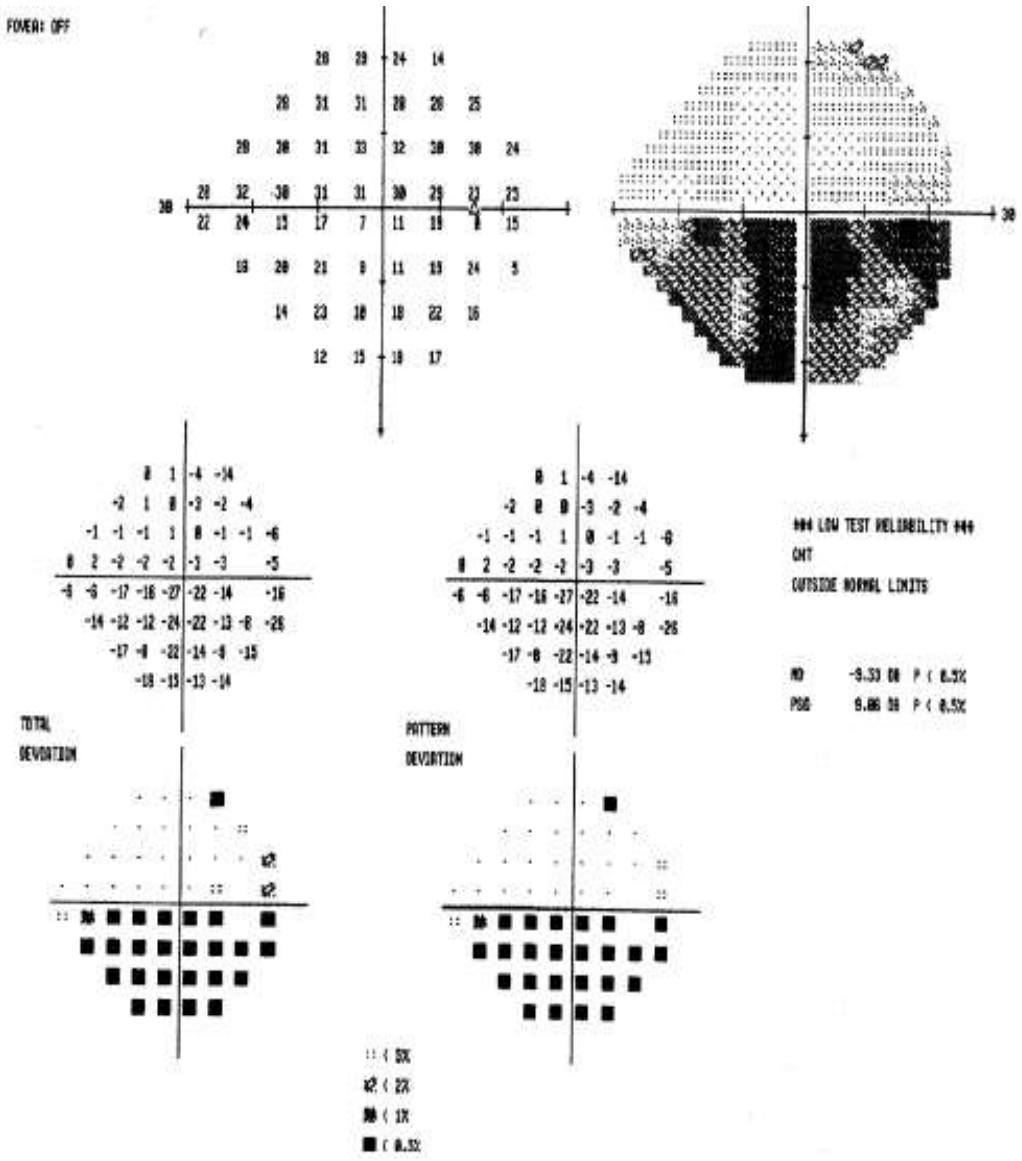


Figure 12- AP showing inferior altitudinal field defect typical of NA-AION

ARTERITIC AND NON ARTERITIC AION

ARTERITIC & NON ARTERITIC AION

FEATURES	NA-AION	AAION
Age	50 years and above usually	60 years and above usually
Headache, claudication, scalp tenderness, temporal artery induration	Less common	More common
Transient visual loss	Rare	Common
Visual acuity	6/60 or better usually	Worse than 6/60 usually
Fundus- 1. Optic disc edema 2. Fellow eye -optic disc 3. After 4-8 weeks	Hyperemic/ pallid Diffuse/ segmental "Disc at risk" Optic atrophy	Pallid and segmental- more common Normal Cup disc ratio Optic atrophy with cupping
FFA	Only disc delay	Choroidal delay with disc delay
ESR	Less than 50 usually	More than 70 usually
Contralateral eye involvement	14%	54-95%
Visual acuity recovery	Stable/ progressive/ recovery	Recovery unusual

DIFFERENTIAL DIAGNOSIS

DIFFERENTIAL DIAGNOSIS

FEATURES	NA-AION	OPTIC NEURITIS
Age	usually 50 years and above	Usually young
Pain with eye movement	Absent	present
Optic disc edema	Diffuse / segmental Hyperemic/ pallid	Diffuse and hyperemic without hemorrhages
FFA	Disc delay	Normal filling of disc
MRI brain	Usually normal	Optic nerve swelling/ enhancement is common

FEATURES	NA-AION	DISC EDEMA DUE TO COMPRESSIVE LESIONS
Visual acuity loss	Relatively rapid	Insiduous onset, gradually progressive
Orbital disease with proptosis, lid or EOM involvemnt	Absent	Can be present
After 4-6 weeks	Atrophy develops	Disc edema persists

FEATURES	NA-AION	DIABETIC PAPILLOPATHY
H/O Diabetes mellitus	Not always	70%- type 1 DM, 30% type 2 DM
Optic disc edema	Usually unilateral	Bilateral in 40%
Visual acuity loss	6/60 or better	6/12 or better if not a/w maculopathy
Optic nerve dysfunction	Present	Absent

MANAGEMENT

MANAGEMENT

INVESTIGATIONS

FEATURES	INVESTIGATIONS
Typical presentation of NA-AION with no symptoms/signs of GCA and normal ESR and CRP	Control of DM, HTN, Hyperlipidemia
Atypical course with pain, persistent disc edema for more than 2 months, progressive visual loss beyond 2 months, recurrence after 2 months	Neuroimaging
Orbital ischaemia	Carotid doppler
Age less than 50	Serum Homocysteine
Clinical features of thrombosis/ vasculitis	Work up for pro thrombotic conditions and vasculitis

CLINICAL COURSE

VISUAL ACUITY

Untreated NA-AION	Usually stable
Untreated NA-AION (progressive form)	Worsens in 1-2 months, with no further deterioration
Untreated NA-AION (spontaneous recovery)	13-42% cases
Recurrence in affected eye after 2 months	3 % cases

VISUAL FIELDS

Improvement in mean sensitivity or retinal threshold sensitivity by atleast 2dB	24% cases

OPTIC NERVE HEAD

Optic disc edema subsides in 4-6 weeks	Diffuse / sectoral atrophy develops
Persistent disc edema after 6 weeks	Alternate diagnosis to be thought of

ROLE OF ORAL CORTICOSTEROIDS IN NON ARTERITIC -ANTERIOR ISCHAEMIC OPTIC NEUROPATHY

The major pathology is stasis of axoplasm due to ischaemia that clinically presents as optic disc edema. As a secondary change the axonal swelling occuring in a crowded disc causes compression of capillaries and fluid leakage. This aggravates the ischaemia forming a vicious cycle[1.]

Corticosteroids break this vicious cycle and cause faster optic disc edema resolution. They reduce the capillary permeability and cause progressive decrease of compression of capillaries in the ONH[2]. Thus they improve the functioning of the surviving but non functioning hypoxic axons. Visual acuity and visual fields improve upto 6 months if

oral corticosteroid therapy is started within few weeks of onset of NA-AION[13].

The following modalities of treatment that were tried for NA-AION were found to be ineffective[3].

- Optic nerve sheath decompression
- Aspirin
- Levodopa/ carbidopa
- Brimonidine
- Hyperbaric oxygen

PREVENTION

Though there is no proven prophylactic measure for NA-AION, Aspirin is recommended in NA-AION for its role in reducing the risk for stroke and myocardial infarction.

PART II

AIM AND OBJECTIVES

AIM AND OBJECTIVES

PRIMARY OBJECTIVE:

To assess improvement in visual acuity and visual fields following treatment with oral corticosteroids in NA-AION during the acute phase

SECONDARY OBJECTIVE:

To assess the rate of resolution of optic disc edema in NA-AION following treatment with oral corticosteroids

MATERIAL AND METHODS

MATERIALS AND METHODS

STUDY CENTRE: Neuro-ophthalmology services, RIOGOH,Chennai

STUDY DESIGN: Prospective study

INCLUSION CRITERIA:

Patients in the age group of 50 years and above of either sex presenting with

- Sudden onset painless defective vision (presenting within 2 weeks of onset)
- Visual acuity worse than 6/12
- Optic disc edema (suggestive of acute phase of NA-AION) which can be hyperaemic/pallid, diffuse/segmental with or without splinter haemorrhages, small or absent cup in either eyes
- Visual fields showing altitudinal/sectoral/ arcuate defects
- FFA showing disc delay without choroidal delay

EXCLUSION CRITERIA:

- Transient obscuration of vision/claudication
- ESR >50mm/hour
- Scalp tenderness or induration/cordlike firmness/ nodularity of temporal artery region
- Recurrent NA-AION in the same eye
- Bilateral NA-AION
- Patients who had a diagnosis of glaucoma & visual field loss
- Patients who are having DM with RBS >140 mgs/dl or diabetic retinopathy other than mild non proliferative diabetic retinopathy
- Patients with immunocompromised status
- Peptic ulcer disease
- Pregnant females
- Presence of infectious /inflammatory disease that could be responsible for optic disc edema
- Presence of other ocular disorders which could have influenced the vision

SAMPLE SIZE: 40 patients

SCREENING PROCEDURES / VISITS:

40 patients were diagnosed as NA-AION in acute phase.

At the first visit-

- Detailed history of present and past illness,drug and treatment H/O-Diabetes, Hypertension, Hyperlipidemia, Migraine, long term drug intake like amiodarone/alpha interferon
- V/A using Snellen's acuity chart
- Pupillary reaction
- Colour vision
- Dilatation and fundus examination
- Visual fields using Automated perimetry or Bjerrum screen
- Fundus photography, FFA
- ESR
- Random blood sugar, complete hemogram, fasting lipid profile, Complete systemic evaluation by cardiologist and physician to rule out systemic association

METHODS:

20 patients among them were randomly selected for steroid therapy with 60mg oral prednisolone once daily for 2 weeks, thereafter tapered by 5mg every 5 days to 40mg either till the disc shows no edema or upto a maximum of 2 months and then rapidly tapered off. 20 other patients were treated with placebo (oral vitamin C 200 mg once daily)

Informed consent was obtained from all patients

FOLLOW UP PROCEDURES / VISITS:

- Assesment of visual acuity,pupillary reaction and fundus examination every week.
- Assessment of visual acuity and visual fields after completion of therapy and at the end of 6 months from the onset of symptoms
- Adverse effects of corticosteroids were looked for.

ASSESSMENT OF PARAMETERS:

- Visual acuity
- Visual fields
- Optic disc edema

RESULTS AND ANALYSIS

RESULTS AND ANALYSIS

The study included 40 patients of NA-AION within two weeks of onset of symptoms

Table 1: Number of cases

Group	Number of cases
Steroid treated group	20
Control group	20
Total	40

DEMOGRAPHIC DETAILS

1.AGE

All patients included in the study belong to the age group of 50 years and above

Mean age in steroid treated group was 54.3 years

Mean age in control group was 53.9 years

2. SEX

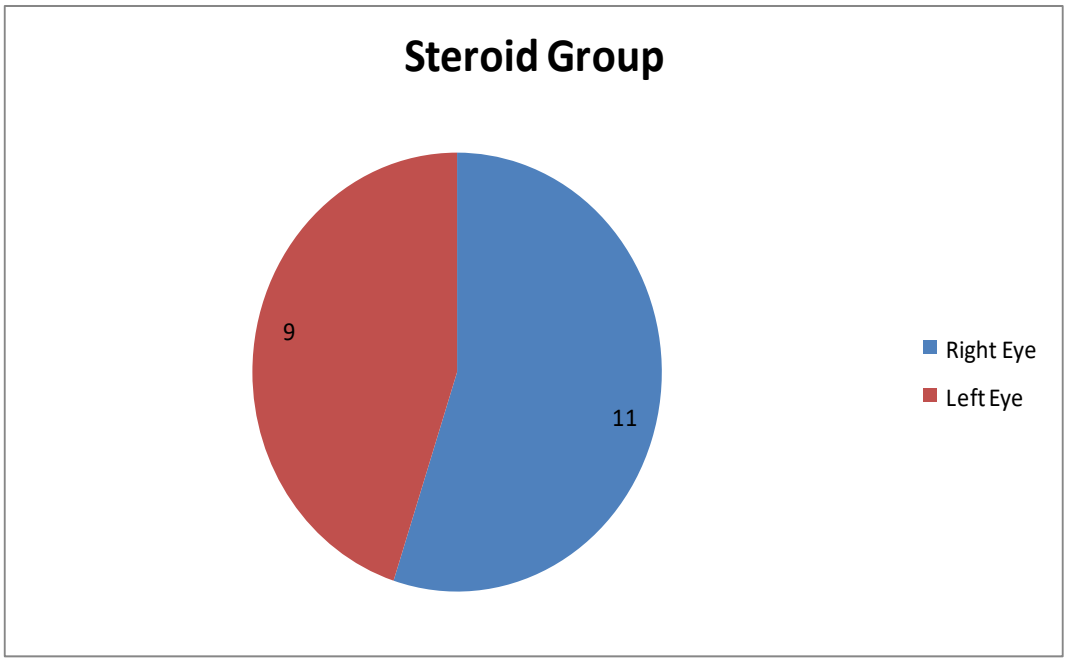
Males were more commonly involved than females.

Table 2: Sex distribution

Sex	Steroid treated group	Control group
Male	13	11
Female	7	9
Total	20	20

3. LATERALITY

Chart 1: Laterality in steroid group



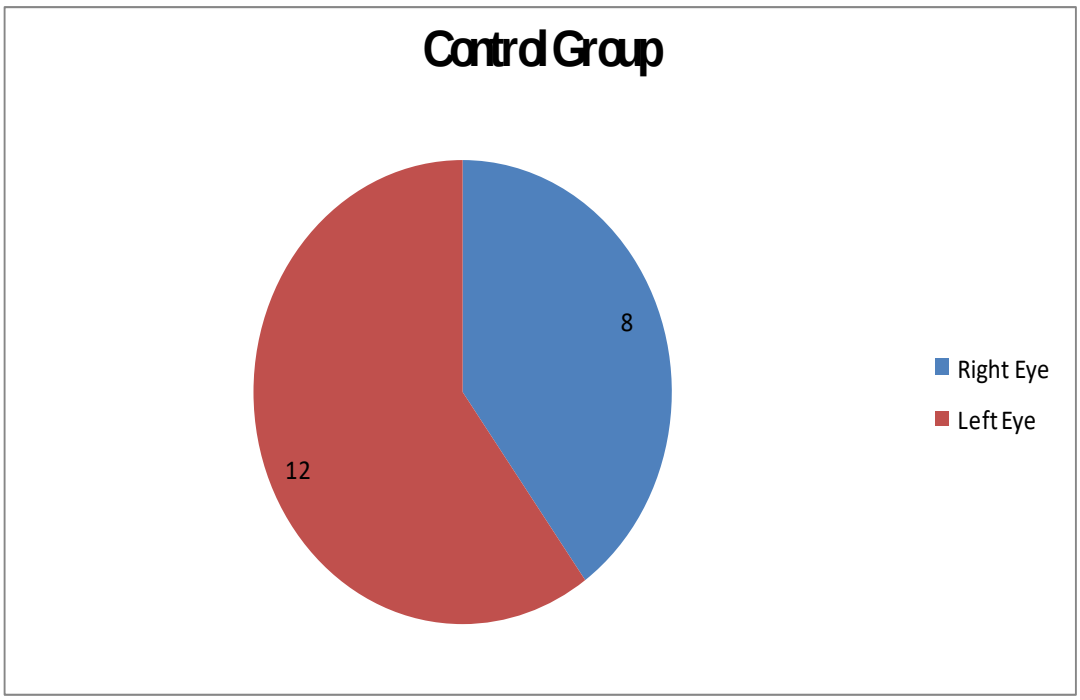


Chart 2: Laterality in control group

3. SYSTEMIC ASSOCIATIONS

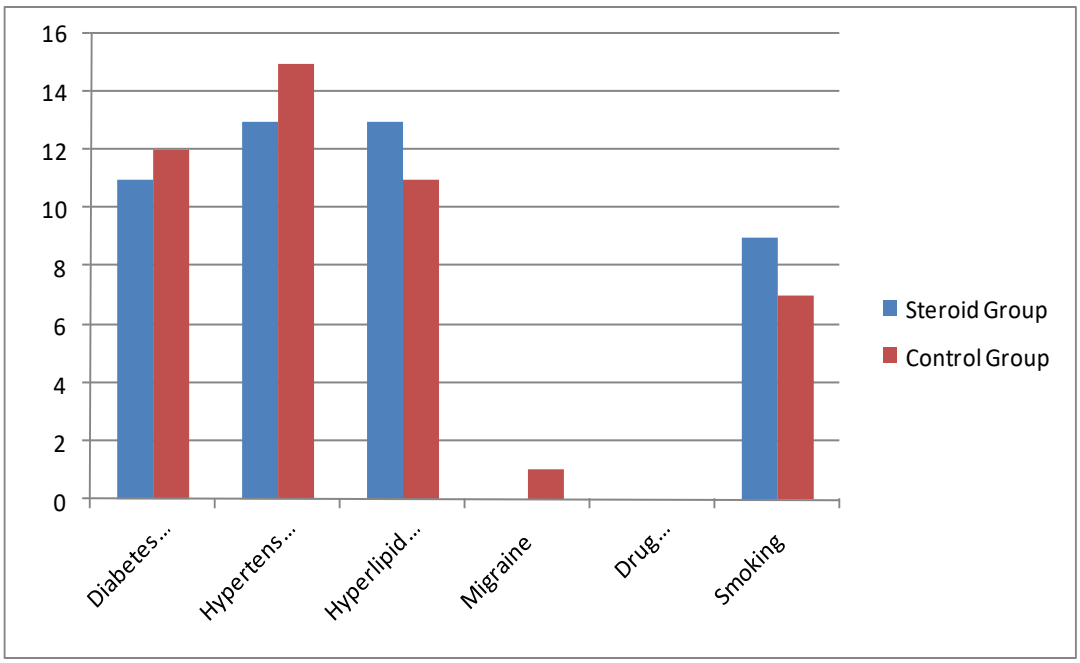


Chart 3: Systemic associations in steroid and control groups

CLINICAL DETAILS

1. OPTIC DISC EDEMA

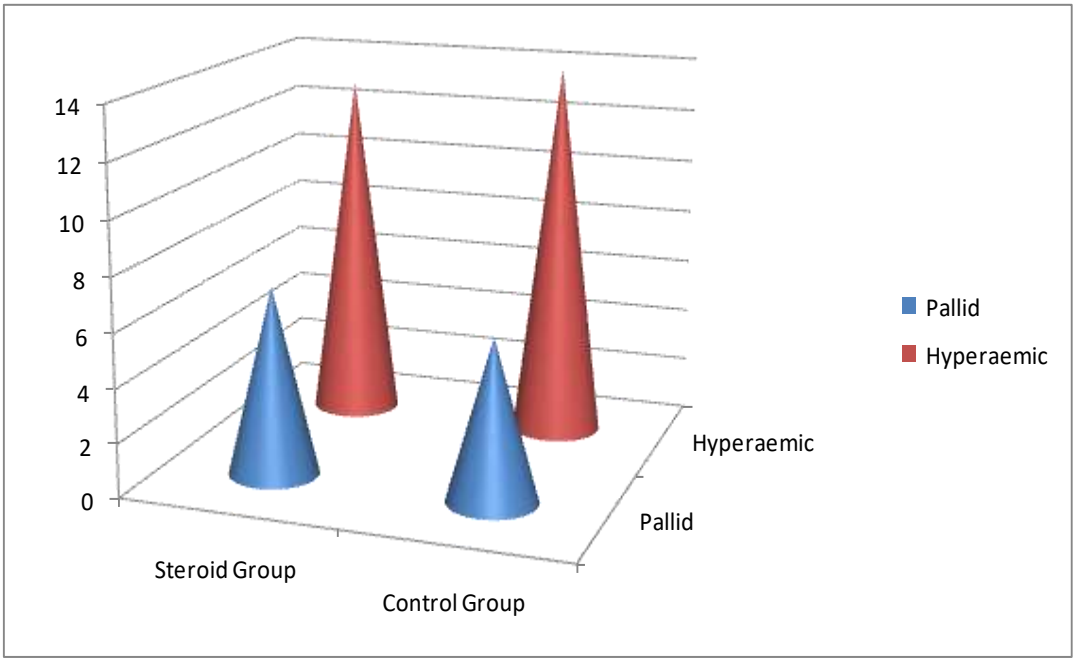


Chart 5: Hyperaemic and Pallid forms of disc edema

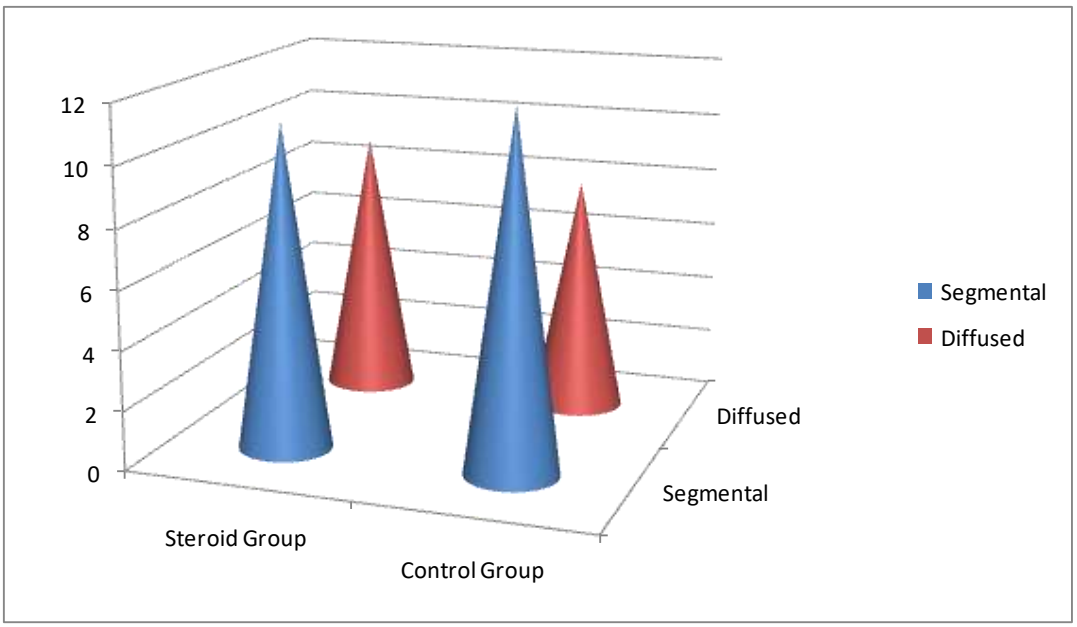


Chart 6: Segmental and diffuse forms of disc edema

Table 3: Fundus in NA-AION

Feature	Steroid treated group	Control group
Splinter hemorrhages	9	7
Small/ absent cup either eyes	14	12

2. VISUAL FIELD DEFECT

Table 4: Type of visual field defect

Type of defect	Steroid treated group	Control group
Altitudinal	8	15
Sectoral	2	0
Arcuate	0	0

VISUAL ACUITY

Improvement / worsening- change in minimum two snellen lines

Visual acuity change from initial visit to completion of therapy						
V/A at initial visit	**Steroid treated group**			**Control group**		
	number of eyes	**improved**	**worsened**	**number of eyes**	**improved**	**worsened**
6/18-6/60	5	4	0	3	1	2
6/60-3/60	3	2	0	5	2	2
3/60-CF	6	3	0	7	1	3
CF or worse	6	3	0	5	0	0
Total	20	12	0	20	4	7

Visual acuity change from initial visit to 6 months from onset of disease						
V/A at initial visit	**Steroid treated group**			**Control group**		
	number of eyes	**improved**	**worsened**	**number of eyes**	**improved**	**worsened**
6/18-6/60	5	5	0	3	1	2
6/60-3/60	3	3	0	5	2	3
3/60-CF	6	5	0	7	2	3
CF or worse	6	3	0	5	1	1
Total	20	16	0	20	6	9

There was significant improvement in V/A in steroid treated group than in control group- p value 0.001

VISUAL FIELDS

Improvement in MS & retinal threshold sensitivity in Automated perimetry or reduction in the size of scotoma in manual fields- improvement

Reduction in MS in Automated perimetry or expanding scotoma in manual fields- worsening

Visual field defect change from initial visit to completion of therapy					
Steroid treated group			**Control group**		
number of eyes	**improved**	**worsened**	**number of eyes**	**improved**	**worsened**
20	7	1	20	3	8

Visual field defect change from initial visit to 6 months from onset of disease					
Steroid treated group			**Control group**		
number of eyes	**improved**	**worsened**	**number of eyes**	**improved**	**worsened**
20	8	1	20	6	9

There was significant improvement in visual field in steroid treated group than in control group- p value 0.01

OPTIC DISC EDEMA

Early optic disc edema resolution - resolution in <6 weeks

Optic disc edema resolution		
Parameter	**Steroid treated group**	**Control group**
Mean duration (weeks)	4.6	7.5
Early ODE resolution-number of eyes (%)	14 (70%)	4 (20%)

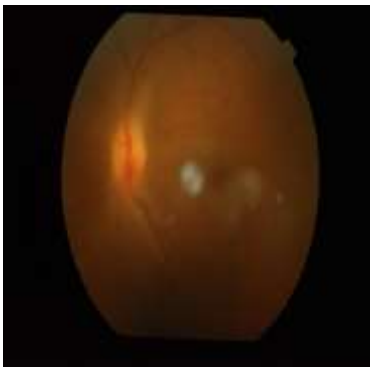
STATISTICAL ANALYSIS

The data collected from all cases were recorded in a Master Chart. Data analysis was done with “Microsoft excel”. The results were analysed with Chi-square tests and p values were calculated. A p value of less than 0.05 is significant.

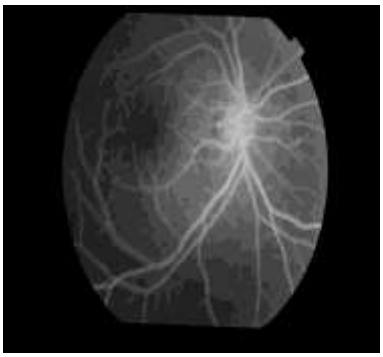
CLINICAL PHOTOGRAPHS

OD- normal

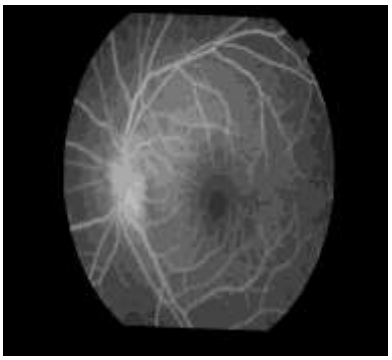
OS- diffuse pallid disc edema



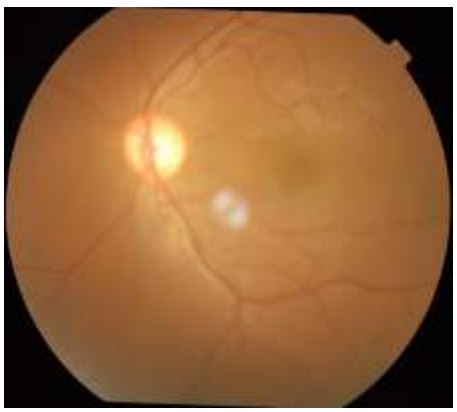
FFA- OD- normal

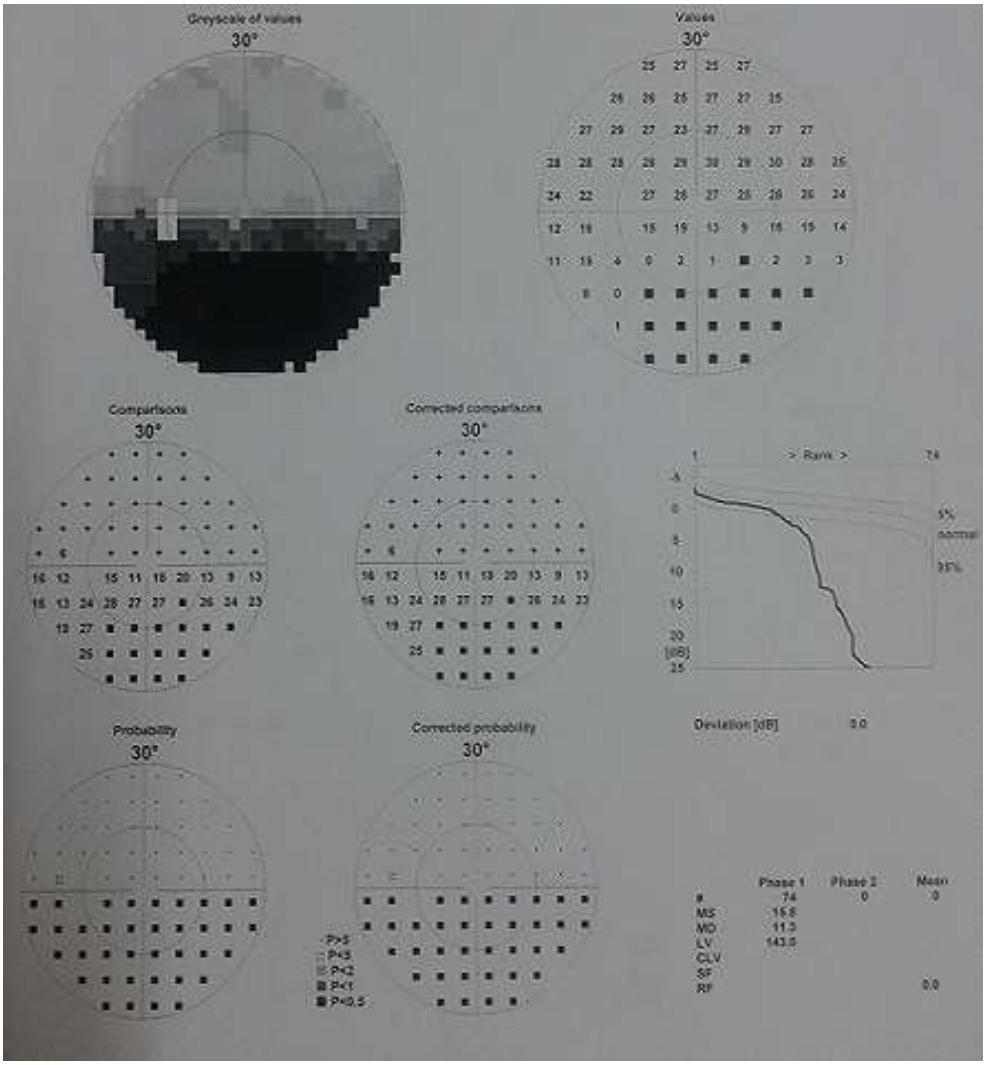


FFA-OS- disc leak



OS- after 6 months- disc pallor

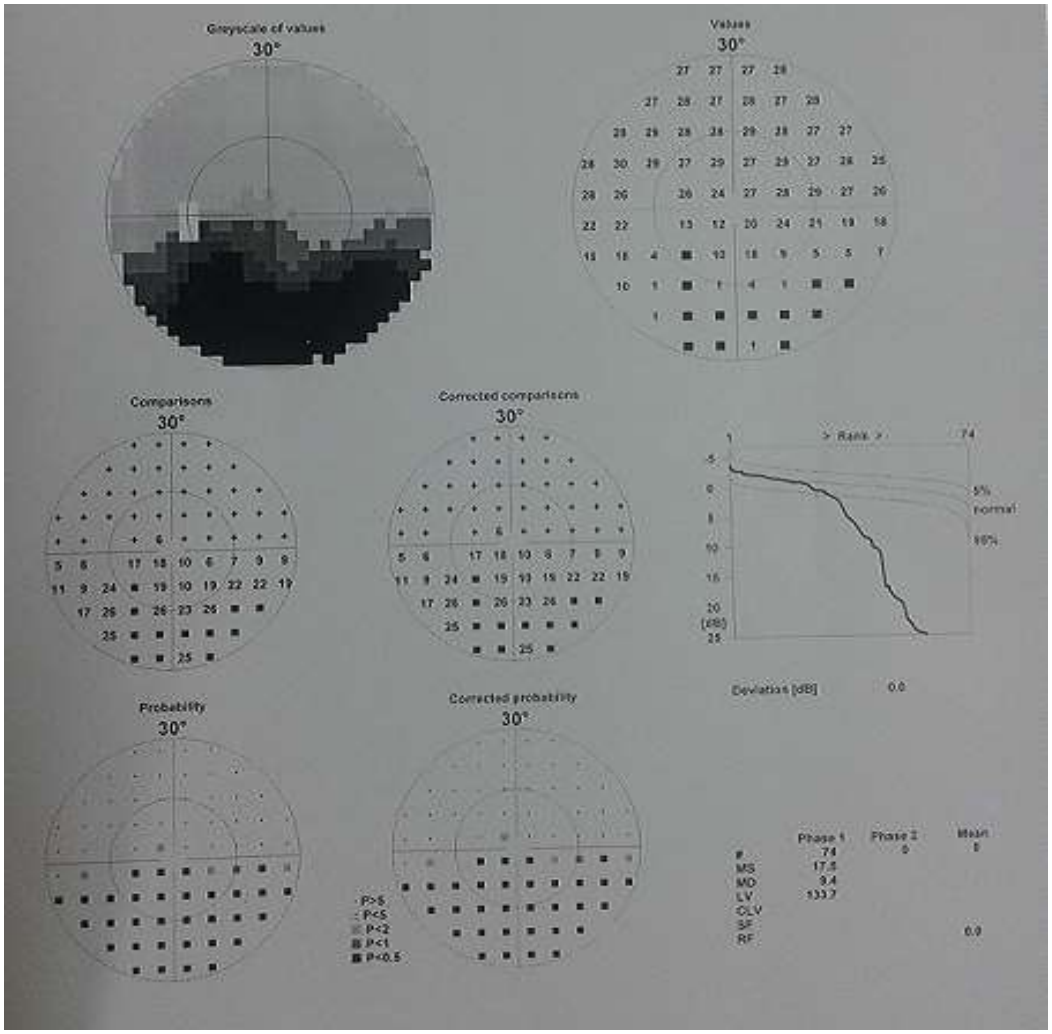




AP OS- BEFORE TREATMENT- INFERIOR ALTITUDINAL FIELD DEFECT

MS-15.8

LV-143



AP- OS – AFTER TREATMENT- INFERIOR ALTITUDINAL FIELD DEFECT – improvement in retinal threshold sensitivity is noted.

MS- 17.5

LV- 133

DISCUSSION

DISCUSSION

Though there are studies which conclude that there is no effective treatment for NA-AION, the role of corticosteroids in it has been studied since 1960s. This study was done to determine whether this therapy is beneficial in NA-AION patients in the early stage of presentation. By analysing our results we find the following:

1. Males are more commonly affected than females.

2. Diabetes mellitus, hypertension and hyperlipidemia were the common systemic associations observed. Smoking as an association was also observed in both the groups. But none of these associations were statistically significant.

3. Relative afferent pupillary defect was observed in 75% of the cases in both the groups.

4. Colour vision defect was observed in 90% of the cases in both the groups

5. The most common type of optic disc edema noted was hyperaemic and segmental type. Small or absent cup in the contralateral eye was seen in more than 50% of cases in both the groups

6. FFA showed disc delay in 50% cases in both the groups and disc leakage was observed in 30% cases

7. The most common type of visual field defect observed was altitudinal especially inferior altitudinal.

8. A change in minimum of two Snellen lines was taken as improvement or worsening. There was 80% improvement in the steroid treated group and 30% in the control group. Irrespesctive of the visual acuity at onset, the improvement in steroid treated group was significant. None of the cases in the steroid treated group showed any worsening in visual acuity. The improvement in visual acuity was noted not only until completion of therapy, but even upto 6 months from the onset of NA-AION.

9. Similarly, the visual fields improved beyond completion of therapy upto 6 months. The retinal threshold sensitivity and mean sensitivity improved after treatment in the steroid group. It improved in 40% in the steroid treated group and 30% in the control group.

10. Early disc edema resolution was noted in 70% cases in the steroid treated group and 20% in the control group. The average time taken for resolution of disc edema was 4.6 weeks in steroid treated group and 7.5 weeks in control group.

11. Corticosteroid therapy did not have any deleterious effects on the visual acuity or visual fields. No serious systemic side effects were observed in any of the cases.

CONCLUSION

CONCLUSION

Oral corticosteroids if started within two weeks of onset of NA-AION is beneficial as it improves the visual acuity and visual fields and hastens the resolution of optic disc edema.

LIMITATION

LIMITATION

The strengths of this study are that it has a control group for comparison and randomization was followed during selection of groups. Though the effectiveness of corticosteroids has been proved in this study, it needs to be extended to a larger cohort.

PART III

ABBREVIATION

- AION- Anterior Ischaemic Optic Neuropathy
- PION- Posterior Ischaemic Optic Neuropathy
- NA-AION- Non-Arteritic Anterior Ischaemic Optic Neuropathy
- PCA- Posterior Ciliary Arteries
- SPCA- Short Posterior Ciliary Arteries
- ONH- Optic Nerve Head
- BP- Blood Pressure
- IOP- Intra Ocular Pressure
- ACL- Anti Cardio Lipin
- A-AION- Arteritic Anterior Ischaemic Optic Neuropathy
- AP- Automated Perimetry
- FFA- Fundus Fluorescein Angiography
- ESR- Erythrocyte Sedimentation Rate

- EOM- Extra Ocular Muscle
- DM- Diabetes Mellitus
- HTN- Hypertension
- GCA- Giant Cell Arteritis
- CRP- C Reactive Protein
- RBS- Random Blood Sugar
- CF- Counting Fingers
- V/A- Visual acuity
- MS- Mean Sensitivity
- LV- Loss Variance
- ODE- Optic Disc Edema

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PROFORMA

Name: Age: Sex:

Occupation: Address:

Registration Number: Contact number:

Presenting complaints:

History of present illness:

H/O defective vision: onset, progression, painful/painless,

H/O defective field of vision

H/O transient obscuration of vision & duration

H/O headache- onset, progression, location, severity, diurnal variation, associated aura/blurring of vision/nausea/vomitting

H/O claudication- jaw,neck or tongue

Past history:

H/O similar episodes in the past

H/O Glaucoma

H/O Diabetes, Hypertension, Hyperlipidemia, Cerebrovascular accident, Myocardial infarction, Migraine

H/O long term drug intake like amiodarone/ interferon alpha

Personal history:

H/O Smoking, H/O alcoholism

GENERAL EXAMINATION

Vital data - pulse rate, blood pressure , peripheral pulses

OCULAR EXAMINATION

Induration of temporal artery region, decreased or absent temporal artery pulse, any cordlike firmness/nodularity of temporal artery, scalp tenderness -Yes/No

Parameters	Right eye	Left eye
Best corrected Visual acuity using Snellen's chart		
Extra ocular movements- ductions and versions		
Anterior segment examination with slit lamp		

Pupil size, shape and reaction		
A dilated fundus examination with slit lamp biomicroscopy using 90D lens and Indirect Ophthalmoscopy using 20D lens- 1.optic disc edema hyperaemic/ pallid , diffuse/segmental 2.Splinter haemorrhages 3.Small or absent cup in either eyes		
Intra ocular pressure measurement by Applanation tonometry		
Colour vision		
Visual fields using automated perimetry by Octopus or Bjerrum screen (if visual acuity is poor)- 1.Altitudinal defect/ Sectoral defect/ Arcuate defect 2. Mean sensitivity		
Fundus flourescein angiography- 1.Disc delay 2.Choroidal delay		

INVESTIGATIONS:

Blood sugar

Complete haemogram with ESR / C- reactive protein

Fasting Lipid profile

Complete systemic evaluation by General physician, Cardiologist to rule out systemic association

DIAGNOSIS-Non Arteritic Anterior Ischaemic Optic Neuropathy

TREATMENT- Oral Prednisolone 1mg/kg body weight once daily tapered by 5mg every week either till the disc shows no edema or upto a maximum period of 2 months

FOLLOW-UP: Every week until completion of therapy & at 6 months fromthe onset of disease. Assessment of

- ✓ Visual acuity
- ✓ Visual fields
- ✓ Rate of resolution of optic disc edema

MASTER CHART

Sr. No	Name	Age	sex	eye	V/A	Systemic conditions	pupil	Colour vision	FUNDUS	AP	FFA	V/A	Field	ODE resolution	V/A	field
1	Samraj	51	M	L	6/36	-	RAPD	Def	P,S,sh	IA	DD	6/18	IA	3	6/18	IA
2	Loganathan	59	M	R	6/36	HTN,HL	RAPD	Def	P,S,sc	IA	DD	6/18	IA	3	6/18	IA
3	Varadaraj	55	F	R	6/36	HTN,HL	RAPD	Def	H,S,sh,sc	IA	DL	6/18	IA	4	6/18	IA
4	Karunakaran	59	M	L	6/60	DM,HTN,HL,smk	RAPD	Def	P,S,sc	IA	DD	6/36	IA	7	6/24	IA
5	Shanthamma	50	M	R	1/60	DM,HTN,HL	RAPD	Def	H,D,sc	IA	DD	2/60	IA	5	3/60	IA
6	Rani	58	F	L	2/60	DM,HTN,HL	RAPD	Def	H,S,sh	IA	DD	2/60	IA	4	3/60	IA
7	Sulochana	51	F	R	6/60	DM,HTN,HL	RTL	N	P,S,sh	IA	DD	5/60	IA	6	2/60	IA
8	Ravi	54	M	R	3/60	HTN,smk	RAPD	Def	P,S,sh,sc	IA	DD	5/60	IA	4	5/60	IA
9	Mohammed ali	55	M	L	CF	DM,HL,smk	RAPD	Def	H,S,sh	NP	DD	CF	NP	8	CF	NP
10	Daniel	53	M	L	CF	DM,HTN,smk	RAPD	Def	H,S	NP	NP	CF	NP	9	CF	NP
11	Krishnan	50	M	R	HM	HTN	RAPD	Def	P,S,sh	NP	DD	HM	NP	9	HM	NP
12	Maniraj	51	M	L	1/60	HL,smk	RTL	N	P,S,sh	IA	DD	3/60	IA	4	4/60	IA
13	Revathi	57	F	R	6/36	DM,HL	RTL	N	H,D,sc	SA	DD	6/36	SA	6	6/18	SA

Sr. No	Name	Age	sex	eye	V/A	Systemic conditions	pupil	Colour vision	FUNDUS	AP	FFA	V/A	Field	ODE resolution	V/A	field
14	Kumar	52	M	L	2/60	DM,HTN,HL,smk	RAPD	Def	P,S,sc	IA	NP	4/60	IA	4	5/60	IA
15	Gomathi	54	F	L	6/36	DM,HTN,HL	RAPD	Def	H,D,sc	IA	DD	6/24	IA	5	6/18	IA
16	Premkumar	53	M	L	CF	HTN,smk	RAPD	Def	H,S,sc	NP	DD	CF	NP	9	CF	NP
17	Kumari	50	F	L	HM	DM,HTN,HL	RTL	N	H,S,sc	IA	DL	HM	IA	5	HM	IA
18	Lakshmi	60	F	L	6/60	HTN,HL	RAPD	Def	P,S,sh,sc	IA	DD	5/60	IA	4	3/60	IA
19	Velmurugan	55	M	L	4/60	DM,smk	RAPD	Def	H,D	NP	DL	4/60	NP	8	4/60	NP
20	Haridoss	52	M	R	HM	DM,HTN	RAPD	Def	H,D	NP	DD	HM	NP	9	2/60	NP
21	Balan	57	M	R	5/60	DM,HTN,HL,smk	RAPD	Def	P,S,sc	NP	DD	5/60	NP	9	5/60	NP
22	Justin	54	M	L	4/60	-	RTL	Def	H,D	IA	DD	4/60	IA	4	4/60	IA
23	Prabakaran	55	M	L	CF	HTN,smk	RTL	Def	H,D,sh,sc	IA	DL	CF	IA	6	HM	IA
24	Muthulakshmi	53	F	L	HM	DM,HTN,HL	RAPD	Def	P,S,sh	IA	NP	HM	IA	5	HM	IA
25	Visalaakshi	56	F	L	CF	DM,HTN.HL	RTL	Def	H,D,sc	IA	NP	3/60	IA	7	3/60	IA
26	Sundar	52	M	L	4/60	DM,HTN,HL,smk	RAPD	Def	H,D	NP	DD	4/60	NP	8	5/60	NP
27	Ananthi	58	F	R	3/60	-	RAPD	Def	P,S,sh	NP	DD	5/60	NP	8	6/60	NP
28	Gopal	55	M	R	CF	DM,HTN,HL,smk	RAPD	Def	H,D,sc	IA	DL	2/60	IA	4	2/60	IA

Sr. No	Name	Age	sex	eye	V/A	Systemic conditions	pupil	Colour vision	FUNDUS	AP	FFA	V/A	Field	ODE resolution	V/A	field
29	Kumaran	50	M	R	4/60	DM,HTN,smk	RTL	Def	H,D,sh,sc	IA	NP	4/60	IA	6	6/60	IA
30	Rosy	54	F	R	1/60	-	RAPD	Def	H,D,sh,sc	Sec	DD	3/60	Sec	5	4/60	Sec
31	Subbamma	56	F	L	4/60	DM,HTN,HL	RAPD	Def	H,S,sc	NP	DL	4/60	NP	8	6/60	NP
32	Palanimuthu	60	M	R	5/60	DM,HTN,HL	RAPD	Def	H,D,sc	NP	NP	3/60	NP	7	2/60	NP
33	Kuppan	52	M	L	5/60	-	RAPD	Def	H,D,sc	Sec	DL	4/60	Sec	5	1/60	Sec
34	Kamatchi	58	F	R	2/60	DM,HTN,HL	RAPD	Def	H,S,sc	NP	DD	1/60	NP	9	1/60	NP
35	Seethamma	51	F	L	4/60/	Mig	RAPD	Def	P,S,sh	NP	DL	3/60	NP	7	2/60	NP
36	Jeevan	54	M	R	HM	DM,HTN,smk	RAPD	Def	H,D,sc	NP	NP	1/60	NP	7	3/60	NP
37	Kamali	53	F	R	5/60	HL	RTL	Def	H,D,sc	IA	DD	6/60	IA	6	6/36	IA
38	Rajan	55	M	R	4/60	DM,HTN,HL,smk	RTL	Def	H,S,sc	IA	NP	5/60	IA	4	6/60	IA
39	Rukku	52	F	L	5/60	-	RAPD	Def	H,D,sh,sc	IA	DL	6/60	IA	6	6/36	IA
40	Murugan	50	M	R	4/60	DM,HTN,HL,smk	RAPD	Def	H,S,sc	NP	DL	5/60	NP	9	6/36	NP

KEY TO MASTER CHART

- M-male
- F-female
- L-left eye
- R-right eye
- CF-counting fingers
- HM-hand movements
- DM-diabetes mellitus
- HTN-hypertension
- HL-hyperlipidemia
- Smk-smoking
- Mig-migraine
- RTL-reacting to light
- RAPD-relative afferent pupillary defect
- Def-defective
- N-normal
- P-pallid
- H-hyperaemic
- S-segmental

- D-diffuse
- Sc-small cup
- Sh-splinter hemorrhage
- IA-inferior altitudinal
- SA-superior altitudinal
- Sec-sectoral
- NP-not possible
- DL-disc leak
- DD-disc delay